PostScript

LETTERS

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Severe polyneuropathy complicating active Crohn's disease: rapid response to Infliximab

Treatment with a chimeric antitumour necrosis factor (TNF) antibody (Infliximab) has been shown to be highly efficient for patients with steroid refractory Crohn's disease (CD).[1] However, the mechanism of action remains largely unknown.[2] Recently, a favourable response to Infliximab treatment was demonstrated in some diseases complicating active CD such as acute idiopathic pancreatitis.[3] We report a case of a middle aged female with CD that developed an aggressive form of polyneuritis resistant to corticosteroids.

A 55 year old White female, weighting 68 kg, presented with exacerbation of CD (CD activity index (CDAI) >450) associated with an aggressive form of polyneuritis involving the right arm and leg with arthralgias, myalgias, and functional impotence. She had been suffering from refractory severe CD involving the ileum and right colon for 10 years and she was taking oral corticosteroids for two years continually with signs and symptoms of chronic corticosteroid abuse. Immunosuppressive therapy with azathioprine was rapidly stopped for gastric intolerance. Neuropathy was characterised by arthralgias, myalgias, and functional impotence of the right arm and leg. Severe muscle atrophy of the right arm was evident and deambulation was very difficult. Therapy with folate, vitamin B12, and vitamin B6 was ineffective. A magnetic resonance scan of the cranium and spinal cord excluded central, optic nerve, and spinal cord demyelinating lesions. Electromyography showed demyelinating neuropathy involving the right and left esternal popliteus ischiatic nerve, a mixed (motor and sensitive) neuropathy involving the right and left radial nerve, and an axonal neuropathy involving the right ulnar and medial nerve. Other conditions such as polyarteritis nodosa, Wegener's granulomatosis, primary mixed cryoglobulinaemia, rheumatoid arthritis, systemic lupus erythematous, or sarcoidosis had been excluded.

An infusion of 5 mg/kg Infliximab (Remicade; Schering-Plough SpA) was given at weeks 0, 2, and 6. Five repeated 5 mg/kg Infliximab infusions at eight week intervals were administered. Infliximab was well tolerated and no side effects were recorded. Arthralgias, myalgias, and functional impotence of the right arm and leg progressively improved after the first Infliximab infusion. Muscle atrophy of the right hand improved dramatically two weeks later. Electromyography performed at week 22 after the start of therapy was normal. CDAI score is <150 at this time. Sign and symptoms of chronic corticosteroid therapy rapidly disappeared.

In conclusion, Infliximab may be a suitable therapeutic option in patients with rare extraintestinal manifestations of CD such as severe polyneuritis not responding to conventional therapy.

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Caution with the use of cyclosporin in pregnancy

We read the article by Alstead and Nelson-Piercy with great interest.[1]

We report a case (submitted for publication) of a woman with fulminant ulcerative colitis in the 29th week of pregnancy. Her disease was refractory to steroids, but she refused to have cyclosporin whilst pregnant. She therefore underwent an emergency Caesarean section and was given intravenous cyclosporin post-operatively. After 48 hours of treatment she developed severe hypertension with hypertensive encephalopathy and seizures. Although cyclosporin has been considered to be safe for both mother and foetus, we would like to highlight concerns that it is associated with potentially life threatening side effects. As a result, patients must be counselled thoroughly about the potential morbidity associated with this treatment and monitored closely. We agree with Dr Alstead in that cyclosporin should be used with extreme caution in pregnancy and the postnatal period.

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Coeliac disease: is case finding the correct ethical and logistical approach?

I read with interest the debate pertaining to screening for coeliac disease (CD). Although one can argue that CD fulfils the tenets of any screening programme, however, we do not know the natural history of screen detected patients with CD.

Logistically when would we decide to screen—at what age and how often thereafter? Serological markers may be highly sensitive and specific but the value of these tests decrease when they are used in the general population.

Although the investigational process for population screening and case finding may be the same, there is an important ethical difference between them. If a patient seeks medical help then the physician is attempting to diagnose the underlying condition (for example: patients with CD who present with symptoms of irritable bowel syndrome). This would be classified as case finding and clearly it is the patient who has initiated the consultation and in some sense is consenting for investigation. Conversely, individuals (who are not patients) found to have CD through screening programmes, may have considered themselves as "well" and it is the physician or healthcare system that is identifying them as potentially ill.

We recently performed a primary care based cross sectional study using immunoglobulins, IgA/IgG antigliadin antibodies and endomysial antibodies to initially recognise CD. 1200 volunteers were recruited from January 1999 to June 2001 from 5 general practices in South Yorkshire, UK. Any participant with a positive IgA antigliadin antibody, positive endomysial antibody or only IgG antigliadin antibody in the presence of IgA deficiency was offered a small bowel biopsy to confirm the diagnosis of CD. Twelve new cases of CD were diagnosed from 1200 samples. The prevalence of CD in this primary care population sample is 1% (95% CI 0.4–1.3%).[1] In this screening study, 9/12 diagnosed cases of CD ultimately had symptoms which could be attributed to CD (for example, anaemia or subtle gastrointestinal symptoms). We, and others have demonstrated a delay in the diagnosis of CD—surely the important change in our clinical practice (both in primary and secondary care) is to have a low threshold for case finding.[2–4] If you look for CD you will find it.

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Author's reply

If I interpret Dr Sanders' position correctly, he favours population screening, provided that a case finding approach is applied. His letter gives me the opportunity to expand further on my opinion on the appropriateness for coeliac disease (CD) population screening. As I mentioned in my final remarks and in the summary of my debate, while CD fulfils the criteria for mass screening, currently we lack the evidence based elements to justify a universal screening in European and North American populations. Therefore, my current position does not diverge substantially from that of Dr Sanders. I firmly believe that an "open-minded approach", in which increased awareness and low threshold are applied to populations at risk, is ethical, logistical, and socially acceptable. This attitude was extremely effective in the USA, where the healthcare community had the perception that CD was extremely rare. We have recently subverted this wisdom by showing that the overall prevalence of CD in the USA is 1:133 in not-at-risk groups and between 2%–9% in at-risk groups, so proving that this disease was historically overlooked in the USA.[1] If some reports in the literature indicating that prolonged gluten exposure can lead to increased morbidity[2] and mortality[3] are confirmed, we should be ready to change our attitude and embrace new guidelines for CD mass screening.

Epidemiological data published worldwide suggest that CD is one of the most frequent genetically based chronic diseases of humankind. Therefore, there has been no better time to establish the appropriateness for CD mass screening by performing well designed studies, rather than look in the opposite direction and ignore the problem. If we are not humble enough to embrace this approach to resolve this issue, we will not only be ethically and logistically incorrect, but also morally responsible for a poor outcome of our medical mission.

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Multiple focal nodular hyperplasia of the liver in a patient with prostatic cancer

We read with interest the study of Luciani and colleagues (*Gut* 2002;**50**:877–80) comparing focal nodular hyperplasia (FNH) of the liver in men and women. The major findings of this very large study conducted by an expert team in this field were that mean age at diagnosis was higher in men ($p<0.01$), mean FNH size smaller ($p<0.001$), and surgery more often performed ($p<0.001$) in men ($n=18$) than in women ($n=216$). Interestingly, perhaps because of the relatively small number of men (although very large in terms of the rare occurrence of FNH in men), no cases of multiple FNH were noted in the male population.

We report here a case of multiple FNH in a 74 year old patient with a biopsy proven prostatic cancer. This patient had not received any treatment. He was referred to our liver unit in March 2001 for evaluation of multiple liver masses discovered on abdominal ultrasonography during the staging of his cancer. Bone scintigraphy disclosed no metastases. Liver biochemistry was normal except for a mild increase in gamma glutamyl transferase activity. Prothrombin index was 100%. Serological search for hepatitis B or hepatitis C virus infection was negative. Genetic (haemochromatosis, alpha 1 antitrypsin deficiency) and autoimmune liver diseases were carefully excluded, and alcohol consumption was below 10 g/day. Upper gastrointestinal endoscopy and colonoscopy were normal. Tumour markers of malignant primitive or secondary liver lesions were within the normal range. Liver Doppler ultrasonography disclosed multiple heterogeneous lesions with a hypoechoic pattern and without an arterial signal. Abdominal tomodensitometry before and after contrast enhancement showed multiple lesions with rapid contrast enhancement during the arterial phase. The largest lesion was located between the left liver and segment IV and measured 75 mm.

Because there was no magnetic resonance imaging (MRI) in our centre, ultrasound guided liver biopsy in both tumoral and non-tumoral areas was performed. The diagnosis of typical FNH was made in several of the lesions whereas non-tumoral liver was normal. The patient received hormonal treatment from April 2001. In October 2001, MRI confirmed a diagnosis of FNH with a central stellate area in the largest lesion. In December 2002, he was in good health with unchanged ultrasonography.

This case report is unique in that there were multiple lesions of FNH in a man who had not received any previous treatments or porta-caval shunt. Although from a literature search it is difficult to determine the exact number of men with multiple FNH, the numbers are probably very low. In this report, the discrepancy between normal bone scintigraphy and multiple liver lesions made the diagnosis of liver metastasis probable. Nevertheless, histological examination of several hepatic lesions, retrospective MRI, and outcome made the diagnosis of multiple FNH certain. This report, in common with the large series of Luciani *et al*, indicates that FNH diagnosis may be very difficult in men

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Guidelines for colonoscopic screening in acromegaly are inconsistent with those for other high risk groups

We welcome the joint work of the British Society of Gastroenterology and the Association of Coloproctology of Great Britain and Ireland in commissioning guidelines for colorectal cancer screening in high risk groups (*Gut* 2002;**51**(suppl 5):V13–14). In the absence of direct evidence from randomised trials for most of the groups, the various authors have balanced a wealth of recent genetic and epidemiological evidence estimating an individual's levels of elevated risk against the risks associated with screening. The end result is the recognition that within the label "high risk" there is a spectrum of risks such that colonoscopic screening and surveillance must be tailored accordingly. This avoids the ineffective, costly, and potentially harmful "blanket-type" approach, which formerly prevailed.

However, within the guideline series, there is one exception—screening and surveillance in patients with acromegaly. We and other researchers[1–3] have repeatedly stated that the studies undertaken by the authors of these guidelines have overestimated the risk of colorectal cancer in this patient group. They report a 13–14-fold increase in risk based on colorectal cancer detection rates among acromegalics undergoing colonoscopy at Barts compared with cancer rates from published series of colonoscopic screening in non-acromegalic subjects (see table 3 in Jenkins and Besser[4]). All of these "control" studies are in mixed race US populations and lack data to permit age-sex comparisons. This simply does not rank as a well designed controlled study and the recommendations should not receive grade B status.

In the same manner as relative risks for those with relatives with colorectal cancer are estimated from population based data (summarised in Dunlop[5]), we have argued for a similar approach for patients with acromegaly.[1] Based on three population based studies, we calculated (by fixed effects meta-analysis) a relative risk of 2.0 (95% confidence intervals 1.4–2.9) for colon and rectal cancer combined in acromegalics.[1 2] Considered in terms of absolute risk, with an approximate 2% cumulative risk of colorectal cancer by age 70 years in the general UK population, the estimated risk in acromegalics would be 4%. Notably, the incidence in the Barts series is 4.5% (10 of 222)—not a quarter if the estimate of a 13-fold increase was applied. Acromegalic patients thus have a modest increase in colorectal cancer risk, not a high risk. In first degrees relatives with a strong family history (a high risk group), Dunlop[5] recommends early colonoscopic screening and then wait until 55 years for repeat colonoscopy if initial screening is clear (the majority). How can Jenkins and Fairclough justify early screening and colonoscopy five yearly thereafter in *all* of their acromegalics?

Atkin and Saunders[6] have demonstrated that colonoscopic surveillance following initial screening in the non-acromegalic population is determined primarily by clinicopathological findings. These basic guidelines must also be applied to the acromegalic patient population. Jenkins and Fairclough have stated that elevated serum IGF-I levels may predict for recurrent adenomas in acromegalic patients and "should be offered screening at three year intervals". This is based on data from only eight acromegalic patients with recurrent adenomas[7] and should not replace other well recognised predictors of recurrence.

Looked at in the context of (other high risk) groups, the guidelines for colorectal cancer screening in patients with acromegaly are inconsistent. The aggressive approach to

colonoscopic screening recommended by Jenkins and Fairclough should be seriously questioned.

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Authors' reply

We thank Dr Renehan and colleagues for their comments on our data, which they have also made previously. We do not claim that our data are perfect in all respects but it seems to us, on the basis of the data we have collected in our own series and that of other groups, that patients with acromegaly should be regarded as having a significantly increased risk of colorectal neoplasia. The two contrary studies referred to by Renehan *et al* are his own and that of one other author who relied upon retrospective data acquired more than 50 years ago. These data and those from the population based studies preferred by Renehan suffer from flaws of their own. The morbidity associated with acromegaly has changed in the last 25 years, probably related to the increased survival associated with aggressive and effective treatment of the cardiovascular and metabolic complications of the disease. Our data and those of others show that the prevalence of colonic neoplasia in acromegaly is age dependent. Thus it is only now that patients are surviving long enough to develop this complication, and valid comparative data must therefore be acquired contemporaneously, to take account of the changing pattern of morbidity associated with increased longevity.

We are aware of at least 12 other prospective studies evaluating colonoscopic screening in acromegaly, in addition to our original report from St Bartholomew's Hospital[1]. These include one by Renehan *et al* in which they reported three asymptomatic patients in whom a cancer was detected. Among such studies the optimum comparison must be simultaneous screening of asymptomatic non-acromegalic subjects. A combined comparison of the data from all series using these control groups, none of which involved mixed race US populations, gives a relative risk of colon cancer in acromegaly of 13.4. We think it is prudent to accept the evidence of an increased risk of colon cancer, derived from these clinical observations rather than from theoretical calculations, and to screen acromegalic patients systematically until the current hypothesis is confirmed or refuted. The rarity of acromegaly means that the increase in workload for the majority of individual endoscopy units is likely to be minimal.

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New imaging techniques: promise or passe

I read with great interest the article by Egger and colleagues (*Gut* 2003;**52**:18–23) evaluating laser induced fluorescence endoscopy (LIFE) and methylene blue (MB) directed biopsies for detection of dysplasia in Barrett's oesophagus.

As the authors point out, there have been no fully published studies to date on this much talked about procedure. The authors found that LIFE and MB had limited accuracy, as did standard random biopsy. Although LIFE and MB detected a total of five cases of high grade dyplasia and 11 cases of low grade not seen on four quadrant random biopsy (4QB), they concluded that these methods are "not capable of increasing the diagnostic accuracy or replacing standard four quadrant biopsies".

How could these data lead to this conclusion? The authors discount all but one high grade and seven low grade lesions detected by LIFE or MB because they were "within the 4QB protocol". It was assumed by the authors that these sites would have been biopsied by random techniques had it not already been sampled with AF or MB. Given that the biopsies were standard 7 mm forceps, that dysplasia can be very focally distributed, and the area included within the 4QB covers two linear centimetres, it is difficult to assume that this exact site would have been biopsied with a random technique. This assumption, if incorrect, would result in underestimation of the value of LIFE or MB.

In addition, the authors further discounted the one remaining high grade dysplasia site and four more low grade sites because they occurred in patients with known cancer who presumably would be treated for the cancer regardless. There is little doubt that detection of low or even high grade dysplasia has little relevance if a cancer is already known. The main group of patients where LIFE, MB, and other advanced techniques should be applied are those without macroscopically evident tumours and cancer. Discounting LIFE and MB for this reason may further underestimate its value.

If we do not discount these cases then LIFE and MB appear to compliment 4QB for the detection of dysplasia, with each technique independently detecting dysplastic sites that the other missed.

I agree that LIFE and MB remain controversial and applaud the authors for publication of this first LIFE trial. Given the constraints of the study however, it may be premature to proclaim these techniques incapable. More well conducted studies are clearly needed. The field of imaging technologies is also evolving rapidly and new and better techniques are constantly on the near horizon.

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Transient ischaemic colitis following an aeroplane flight

We read with interest the report of Butcher and colleagues (*Gut* 2002;**51**:746–7) of two cases of transient ischaemic colitis following an aeroplane flight. This report represents more evidence supporting the suggestion of a possible important role of acquired and hereditary thrombotic risk factors in the pathogenesis of ischaemic colitis.[1 2] However, the largest study to date concerning these factors in patients with ischaemic colitis was not mentioned in the study.[1] Moreover, the reported patients may also have other acquired or inherited thombophilic disorders which were not evaluated. Lipoprotein (a), anticardiolipin antibodies, the C677T methylenetetrahydrofolate reductase polymorphism, and the G20210A prothrombin gene mutation were not studied in both patients whereas in the second case even the very important factor V Leiden mutation as well as lupus anticoagulant and homocysteine levels were not evaluated. Although the aeroplane flight could be the most important risk factor in these cases, the rather incomplete thrombophilic screening does not permit us to conclude that it was "the only potential risk factor".

It is known that deep vein thrombosis (mainly symptomless) may occur in up to 10% of long haul airline travellers.[3] In contrast, hypercoagulable states may play an important role in ischaemic colitis, leading to the development of thrombotic occlusion of the small vessels supplying the colon. In a recent study of comprehensive thrombophilic screening in patients with an established diagnosis of ischaemic colitis, we found the prevalence of acquired and hereditary thrombotic risk factors significantly higher compared with the prevalence of these factors in matched inflammatory and healthy controls.[1] A thrombophilic tendency was demonstrated in the majority of patients and the most significant associations were with antiphospholipid antibodies and with the factor V Leiden mutation. Moreover, we recently found a high frequency of protein Z deficiency in patients with ischaemic colitis (unpublished data). Based on the recent data of the association of protein Z deficiency mainly with arterial thrombosis,[4] protein Z deficiency may be involved in the development of the disease in a subgroup of patients by causing thrombosis in the small mesenteric arteries.

Ischaemic colitis is considered the result of localised non-occlusive ischaemia of the small arteries. In contrast, the presence of hypercoagulable states suggests a possible role of

venous obstruction. It is possible that future identification of subgroups in ischaemic colitis patients with sophisticated imaging techniques could distinguish cases with arterial or venous ischaemia.

In conclusion, we suggest that the mechanism of ischaemic colitis is multifactorial. Acquired and genetic factors may interact leading to disease manifestation. Arrhythmia and embolic conditions, oral contraceptives, and other medications, as well long haul flights probably play a role in genetically predisposed individuals in the disease pathogenesis.

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BOOK REVIEWS

Surgery of the Liver, Bile Ducts and Pancreas in Children

Edited by E R Howard, M D Stringer, P M Columbani. London: Arnold, 2002, £125, pp 527. ISBN 0-340-76129-6

This excellent volume builds on the success of the first edition published a decade ago by the senior editor (ERH). Inevitably it is bigger, and, yes, better too. The contributors, drawn from across the world, have been chosen for their expertise and this is apparent in their respective chapters. A major beneficial innovation is inclusion of an editor and other contributors from the USA; sadly, for an inherently English book, English spelling has been sacrificed in the process.

All chapters have been rewritten in response to the significant developments in hepatobiliary surgery over the past decade—for example, in our understanding of the anatomy of the liver and in liver transplantation (now up from 14 to 98 pages). The chapter on "portosystemic shunt surgery" has given way to "surgery for portal hypertension", reflecting the development of alternative strategies for this condition. As before, detailed and informative chapters on anatomy, physiology, and preoperative management complement the core "surgical" chapters, and there are new chapters on embryology, anaesthesia and perioperative care, nutrition, and the psychosocial aspects of childhood liver disease, which will be of interest to gastroenterologists and surgeons alike. Another innovation is the inclusion of a section on "surgical" paediatric pancreatic disorders, including chapters on embryology and anatomy; this is a logical development that enhances the value of this volume.

The scientific standard is high throughout the book. Each of the topics has been well researched by its author and (with only one exception), chapters are well referenced. Of course one can find omissions but these are minor and very infrequent. The text is easy to read and the editors have ensured that the content is clinically relevant. Each chapter concludes with a useful table of annotated key references, and in some chapters a recommended management strategy is summarised in a box. The book is liberally illustrated with line drawings and both black and white and colour figures, which generally are informative and clearly reproduced. The index is good; I was able, without difficulty, to locate a variety of topics. Overall, the quality of the production is excellent, with very few typographical errors. Although this is a comprehensive book, it is not a large tome, but is comfortable and easy to read.

This book will serve as an invaluable resource for gastroenterologists, paediatricians, surgeons, nurses, and others involved in the surgical care of children with liver, biliary, or pancreatic disorders, whether at a specialist children's centre or a district hospital, and will appeal to trainees, whose exposure these days to complex hepatobiliary surgery is limited. In addition, gastroenterologists and surgeons treating the increasing number of adults with disorders of childhood will find this book a most helpful companion.

D Lloyd

Gastrointestinal Mucosal Repair and Experimental Therapeutics

Edited by C-H Cho, J-Y Wang. Karger Press, 2002, pp 252. ISBN 3-8055-7382-0

This book is a worthy addition to the *Frontiers of Gastrointestinal Research* series that focuses on specialised gastrointestinal research topics. This volume is the 25th of the series and highlights areas of current and emerging interest to investigators in the area of gastrointestinal mucosal injury, repair, and therapeutics.

The book is not a compilation of talks given at a conference but rather an honest attempt to inform about specific research areas within that broad field. It has been a number of years since anyone has gathered the information for a reference book for his area, and the editors have intentionally chosen to invite experts to write on their specialised subjects. The emphasis of the book is on the use of experimental cell culture and animal models and, therefore, will be of most use to basic researchers. The book is divided into three broad sections, covering epithelial restitution, mucosal repair and healing, and experimental therapeutics. The chapter authors have taken care to summarise what is known and what remains to investigate. Several of the chapters provide excellent reviews of a particular area, including those on angiogenesis, the diacylglycerol/protein kinase C pathway, nitric oxide and its regulation, the roles of cyclooxygenase inhibition, and the involvement of prostaglandin receptors in the gastrointestinal tract. Other chapters contain useful methodology regarding animal models and experimental techniques such as physical stress and strain. The chapters dealing with cytokines and *Helicobacter pylori* infection are brief, but these subjects are adequately covered elsewhere in numerous reviews. New and potentially useful therapeutic possibilities are examined, including the use of platelets to deliver healing growth factors, the use of polysaccharides such as heparin for gastrointestinal protection, and gene therapy with angiogenic growth factors.

It is not possible for any book to be complete and inclusive of all subjects in such a diverse field as mucosal repair and therapeutics, but this book does a more than reasonable job. It will be a very useful reference for research newcomers and veterans alike in the field.

E J Dial

The Handbook of Clinical Trials and Other Research

A Earl-Slater. Abingdon, UK: Radcliffe Medical Press Ltd, 2002, pp 352. ISBN 1-85775-485-9

Clinicians are notorious for embarking upon research without a full understanding of methodology. Perhaps in the past clinical journals were guilty of publishing papers without being sufficiently critical. No doubt this was a byproduct of well meant refereeing by clinicians who were themselves hamstrung methodologically, and lacking insight.

In the new world of publications, the research design has to be explicit, well laid out, and sufficiently robust to support the research reported. Most doctors have had little or no training in research methods, despite having completed an MD. This might be one reason why it is becoming increasingly difficult for even research experienced clinicians to initiate new projects. Indeed, there is a question mark as to whether research can now be done by service based clinicians or whether, because of the newer strictures and disciplines, this should be left to the professional researcher. Perhaps the answer is that clinicians ought to have access to experts who can not only guide them but see them through the entire project. Some have questioned whether clinical research is still possible—the answer, even if only in hope, is yes, but this will require a greater familiarity with research methodology, the patience to plan thoughtfully, and with experienced counsel.

This handbook provides definitions and contemporary examples. It provides recent references from major journals and is well illustrated. It contains material beyond explanations of research terminology and methodology including the new requirements for the Research Ethics Committees, and the EU Clinical Trials directive. Many of these areas are fast moving; in the UK there is a single electronic ethics application form now with new rules regarding consent for multicentre

studies. The only way to keep up to date with many of these developments is to search the appropriate website.

It is a challenge to create a tool that is expected to be appropriate for people with different levels of knowledge and experience. This publication is certainly straightforward to use and is a useful repository of terms and other sources of information. I am glad I have my copy but can you really teach old dogs new tricks?

P Hungin

Hepatology Principles and Practice

E Kuntz, H-D Kuntz. Berlin: Springer Verlag, colour, pp 825. ISBN 3540 42161 0

"You have a very large parcel", Zeinab, my secretary said breathlessly as she struggled up from the post room with a copy of this enormous book. It seemed all the heavier as I lugged it around the London bus network from the wilds of East Acton on my way home and then back to work several times. Contained in a reinforced Harrod's bag, which was the only thing I could find that was strong enough to hold it while I elbowed my way through the myriads of commuters that were forced to travel by bus in lieu of the non-existent Central line, I felt my back pain had returned with a vengeance and did not know whether to take up weight training (where the book would come in handy) or admit defeat and sue the authors for damages. However, sanity soon prevailed and I soon became engrossed in this weighty tome.

At first glance, one could say that another comprehensive book on hepatology is really not needed, given all the other titles on the market place, but it turns out that this 2002 English edition of the original German "*Praktische Hepatologie*", published in 1998, has been updated and was a labour of love by Erwin Kuntz whose son, Hans-Dieter, died before the book could be finished. My schoolboy German is not up to the original edition but this has become a bible in Germany and the current version is welcome, despite the fact that a lot of the English is somewhat awkward with curious turns of phrase. Examples of this include "MRT", rather than MRI, which is irritating, and "lethality", rather than mortality, which interrupts one's reading pattern. However, there are some very nice things about the book. It has an interesting section on the history of hepatology, perhaps written from a German perspective, since the late Dame Sheila Sherlock may have had a different take on events. Otherwise, there are 40 chapters, which are essentially written as a "hairdresser's guide" to being a hepatologist, with nicely illustrated sections on anatomy (termed "morphology") and biochemistry, before launching into sections on clinical findings and laboratory tests which are comprehensive and well written. There are unusually detailed sections on how to perform liver biopsies and a practical manual on ultrasound (termed "sonography"), the likes of which I have not found in books from the English speaking world, probably because ultrasound is the domain of gastroenterologists in Germany but the preserve of radiologists in the UK. Nevertheless, this level of detail is useful for hepatology/gastroenterology SpRs who would like to interpret scans better but feel inhibited asking their local *x* ray department for fear of looking stupid. However, the section on CT is scanty and on MRI ("MRT") is almost non- existent by comparison. It is a shame that the chapter on cognitive testing and the investigations of the neurological sequela of liver disease does not contain detail on newer psychometric tests and technology such as MR spectroscopy, given the wealth of expertise on hepatic encephalopathy that currently exists in Germany. Chapters on the complications of chronic liver disease are well set out and those on liver abscesses, bacterial, parasitic, and fungal ("mycotic") liver disease are useful.

The question of who may actually buy this book looms large. It is too big and too costly for any individual junior doctor who might be interested in this "user's guide" approach that the book adopts. However, I would have thought given the fact that the format of its main competitor, the Sherlock book, is looking dated by comparison and that other books are not as visually inviting, the Kuntz tome would find a home in most hospital libraries, or if the local gastroenterology department is feeling flush, then on the shelf of the departmental secretary for reference use by all who pass by. For what it is worth, Zeinab has decided to check the strength of her newly reinforced shelves by clearing a space in anticipation of her copy.

S D Taylor-Robinson

NOTICES

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The ACGBI at the Royal College of Surgeons of England, 35–43 Lincoln's Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acpgbi@asgbi.org.uk; website: www.acpgbi.org.uk

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: Torkel.Wadstrom@mmb.lu.se; website: www.helicobacter.org

Falk Symposium 135—Immunological Diseases of Liver and Gut

This symposium will be held on 12–13 September 2003 in Prague, Czech Republic. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

The European Society of Parenteral and Enteral Nutrition (ESPEN)

ESPEN will celebrate its silver anniversary at the time of the annual congress, which is to be held on 20–23 September 2003 in Cannes, France. Further details: www.espen.org

XII Falk Liver Week

The XII Falk Liver Week, in honour of Hans Popper's 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details - see Falk Symposia above.

European Course on Laparoscopic Endoscopy

This course will be held on 18–21 November 2003 in Brussels, Belgium. Further details: Secretary to Professor Cadière, Service de Chirurgie Digestive, Rue Haute 322, Brussels 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)2 647 86 94; email: straeb.asmb@proximedia.be; website: www.straeb-asmb.com

Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is "Liver Diseases in the Post-Genomic Era". Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org